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THE UNIVERSITY OF ALBERTA

CAUSAL ATTRIBUTIONS IN SHOCK-INDUCED AGGRESSION

by

© RUSSELL A. POWELL

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

DEPARTMENT OF PSYCHOLOGY

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THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled "Causal Attributions in Shock-Induced Aggression" submitted by Russell A. Powell in partial fulfilment of the requirements for the degree of Master of Science.

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ABSTRACT

While it has been demonstrated that aversively stimulated animals may attack targets which are only minimally associated with the aversive stimulus (Azrin, Hutchinson, & McLaughlin, 1965; Cole & Parker, 1971), the effects on aggression of strong target-aversive stimulus associations have not been adequately researched. The present study investigated the notion that rats would show a higher rate of shock-induced aggression toward a target with a scent that had been previously conditioned as an excitatory stimulus for shock as opposed to a target with a scent that had been previously conditioned as an inhibitory stimulus for shock. This hypothesis is related to a classical conditioning interpretation of causal attributions in animals (Testa, 1974). Also investigated were two ancillary hypotheses that rats would reliably attack inanimate stuffed-rat targets, and that attacks toward such targets would decrease across sessions. Although the two ancillary hypotheses were supported, the major hypothesis was not. Results are discussed and interpreted with suggestions given for further research.

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Introduction

Aversive stimulation can motivate certain species of animals to exhibit agonistic behaviors toward available targets (e.g. Ulrich & Azrin, 1962). It has been assumed that such pain-induced aggression is of a nondiscriminating nature, such that the stimulated animal is equally likely to attack any suitable target within range (e.g., Azrin, 1967). The purpose of the present study was to test an alternative hypothesis that pain-induced attack is more likely to be directed toward targets which have some degree of close association, or causal relationship, with the aversive stimulation.

The pain-induced aggression phenomenon was first noted by O'Kelley and Steckle in the late 1930s. While attempting to investigate certain effects of electric shock delivered to groups of rats, they incidentally discovered that electrical stimulation of the animals *en masse* resulted in their engaging each other in vigorous and persistent fighting. Some subsequent research was done on the phenomenon, and the results were published in a short article (O'Kelley & Steckle, 1939). In the following years, however, pain-induced aggression received only minor attention in the literature (Daniel, 1943; Miller, 1948), and it was not until the 1960s that the phenomenon was rediscovered during an attempt to negatively reinforce rats to engage in approach behaviors. Azrin (1967, p.116) reported that the intended procedure involved shocking the animals until they approached each other at which point the shock would be turned off. But, like O'Kelley and Steckle, the researchers soon found that the shock stimulation created violent fighting which persistently disrupted their plans. However, in the spirit of Skinner's first principle

of scientific methodology, i.e., "When you run onto something interesting, drop everything else and study it" (Skinner, 1956, p.223), they abandoned their original study and embarked upon the first thorough investigation of the pain-aggression phenomenon. Their first article (Ulrich & Azrin, 1962) marked the cornerstone for all subsequent research in the area.

During the intervening years, a number of articles have been published concerning aggression induced by physically painful stimulation. Shock induction of fighting between paired live rats is still the most common procedure (e.g., Farris, Gideon, & Ulrich, 1970; Lyon & Azolins, 1970; Tondat, 1974; Ulrich & Azrin, 1962). However, other pain-induction methods, such as tail-pinching (e.g., Azrin, Hake, & Hutchinson, 1965) and heat (Ulrich & Azrin, 1962), have been employed. The range of species studied has included ground squirrels (Turner, Boice, & Powers, 1973), squirrel monkeys (e.g., Azrin, Hutchinson, & McLaughlin, 1965), and even turtles and snakes (Azrin, 1967).

An interesting variant of aggression induced by physically aversive stimulation is aggression produced by frustrating, i.e., "psychologically" aversive, stimulation. Azrin, Hutchinson, and Hake (1966) provoked food-deprived pigeons to attack a nearby restrained target bird by extinction of intermittent food rewards. Such aggression has also been induced by increasing time intervals between food presentations (Flory, 1969), and by making food contingent upon difficult schedules of operant responding (Cherek & Pickens, 1970). As with the above research, the majority of frustration-induced aggression studies have employed pigeons as subjects but the effect has also been demonstrated with domestic fowl (e.g., Duncan & Wood-Gush, 1971) and rats (e.g., Gallup, 1965).

It has been assumed by some researchers that aversive stimulation

produces a general tendency to behave aggressively. For example, Scott (1958) contends that pain acts as a primary stimulus for attack. Similarly, Azrin (1967) states that, "...under the stimulus of pain, animals will attack and try to destroy almost any 'attackable' object in the environment....regardless of its attributes" (p. 118). Azrin further reasoned that this response tendency is naturally adaptive: "From the standpoint of evolution, pain-provoked aggression seems to have survival value, since it causes the animal to react instantly and vigorously to noxious events - and in a way that is likely to terminate them" (p. 121).

There is some evidence to support the notion that a general aggressive tendency does result from aversive stimulation. In one experiment, squirrel monkeys were provided with a chain-pulling response which introduced a tennis ball into their chamber. When the monkeys were shocked, they consistently pulled the chain and attacked the ball when it appeared. In the absence of shock, little chain-pulling occurred (Azrin, Hutchinson, & McLaughlin, 1965). In another experiment, pigeons, who were engaged in a difficult and presumably frustrating schedule of operant responding, learned to peck a key to gain access to and attack a restrained target bird (Cole & Parker, 1971). In both examples, the animals sought out and attacked targets which had only minimal associations with the aversive event. It is difficult to construe any motive for the subjects' behavior other than the act of aggression, in and of itself.

On the basis of the above studies, it seems reasonable to conclude that, at least in some instances, aversive stimulation may motivate animals to attack targets that have little association with the aversive event. However, the question remains whether the course of aggression

can be influenced by the presence of a target that is strongly associated with the aversive event. More specifically, it has been anthropomorphically speculated by some researchers that aversively motivated attack is often influenced by the perception of a "causal relationship" between the target and the aversive stimulation. O'Kelley and Steckle (1939) conjectured that a process of "projection" may be occurring during the shock stimulation. In other words, the stimulated rat attacks the other rat because it perceives that rat as being the cause of its pain. Similarly, Dollard, Doob, Miller, Mowrer, and Sears (1939) hypothesized that a frustrated organism will tend to attack "the agent perceived to be the source of frustration" (p.39).

Of direct relevance for the analysis of aggression, both human and animal, some researchers have recently attempted to relate perception of causality to the classical conditioning paradigm. For example, Leger and Rule (Note 1) have argued that causal attributions play a major role in classical conditioning effects on human aggression. The degree to which an attacker becomes a conditioned stimulus (CS) for an aversive assault, is determined by the degree to which responsibility for the assault is attributed to the attacker or to environmental influences.

In the realm of animal learning (though not specifically dealing with aggressive behaviors), Testa (1974) has argued that enhancing the relationship between the CS and the unconditioned stimulus (UCS) facilitates the classical conditioning process, and he related this finding to the acquisition of causal relationships by animals. More specifically, he postulated the existence of "nonspecific associative mechanisms" which are activated by forms of "event covariance". Event covariance is viewed as the defining characteristic of mechanical

causality where one event is perceived as cause because it consistently and reliably covaries with a subsequent event--the effect. By means of the nonspecific associative mechanism, the organism will condition more readily when the CS and UCS covary to a high degree, i.e., when the two events approach a mechanical cause and effect relationship.

Testa (1975) supported his argument by demonstrating instances of conditioning that were enhanced by increasing the similarity between the spatial locations and/or temporal intensity patterns of the CS and the aversive UCS. For example, rats conditioned more readily to a light CS when it had the same spatial orientation as an air blast UCS, i.e., when both events originated from the floor or ceiling. In addition, conditioning was facilitated when light and air blast had the same temporal patterning, e.g., pulsed patterns. Testa (1974) has stated, however, that even the traditional conditioning procedure, involving simple temporal pairing of CS and UCS, can be interpreted within a causal relationship framework. In other words, "temporal contiguity and/or contingencies define two of the characteristics of mechanical causality..."while... similar location and temporal intensity patterns define two more" (p.494). Finally, while Testa dealt mostly with conditioned suppression and avoidance learning, it is implicit within his analysis that his formulation is applicable to other types of conditionable phenomena.

The purpose of the present study was to extend Testa's formulation to the analysis of aversively stimulated aggression in animals--more specifically, shock-induced aggression in rats. It was hypothesized that the aversively stimulated rat demonstrates a higher attack rate toward that target most closely associated with the aversive event, i.e., which most reliably covaries with that event in a CS-UCS

relationship, as opposed to less closely associated targets. Such a response tendency would seem naturally adaptive for an animal's survival since, in at least the natural environment, that which most reliably covaries with and predicts aversive stimulation would most likely be the cause of such stimulation. Thus, while concurring with Azrin that pain-induced aggression probably has functional utility in removing the cause of the aversive stimulation, the present formulation is more specific by predicting that the causal agent will be the preferred target. The pain-provoked animal is thereby endowed with a greater defensive efficiency than if it simply engaged in nondiscriminative aggression.

The present hypothesis actually consists of a reformulation of the aforementioned speculations by Dollard, et al., and O'Kelley and Steckle. However, rather than attempting to deal with mentalistic notions of perceived causal relationships between targets and aversive events, it is instead postulated that a target is attacked because it may be functioning as a CS for the aversive UCS. This latter proposition is not only devoid of unnecessary anthropormorphism, but can also be investigated experimentally.

Ancillary to the main issue, the present study also tested the proposition that an organism can function as a CS. This notion has, to date, received little attention. Timberlake and Grant (1975), in an autoshaping study with rats, used delivery of another rat into the chamber as a CS for food delivery. Reiter and de Vellis (1976) used a human stimulus as a CS to produce conditioned suppression in other humans. Insofar as the possibility that an organism can function as a CS is crucial in attempting to apply classical conditioning to a general analysis of aggression, the present study attempted to replicate the above positive

results within the context of aggressive behaviors.

Finally, because this study employed a unique procedural variant of the shock-induced aggression paradigm, it permitted certain empirical comparisons with results of studies utilizing the more traditional procedure. For example, since models made from stuffed rat-skins were employed as targets in this study, it directly challenged the findings of Ulrich and Azrin (1962) that shocked rats will not attack inanimate targets. Further, within this procedural variant, the present study tested the generally accepted finding that shock-induced aggression is highly persistent across repeated stimulations. Ulrich and Azrin as well as Ulrich and Craine (1964) reported such persistence, but both of these studies employed paired live rats as combatants. Insofar as the present study employed inanimate targets, it removed the possibility of a confounding influence, upon the rate of aggression, of aversive stimulation from a retaliating adversary as opposed to shock stimulation alone. On the basis of pilot data, it was hypothesized that, with inanimate targets, a significant across session decrease in rate of aggression would occur.

Design Overview

The experiment constituted a split-plot design with repeated measures and consisted of two distinct phases: a training phase and a test phase. The training phase was, essentially, a classical conditioning procedure. Subject animals were conditioned to each of two distinctive olfactory stimuli, i.e., scents. One scent was paired with shock sessions to become a conditioned excitatory stimulus (CS+), while the other scent was paired with nonshock sessions to become a conditioned inhibitory stimulus (CS-). The scents were counterbalanced over two groups to each of which subjects

were randomly assigned. Repeated measures of various indices of conditioning were taken across CS+ and CS- sessions for each subject. These indices consisted of drinking rate, grooming rate, and rate of defecation.

When the subjects had demonstrated a significant degree of conditioning to the scent stimuli, the test phase was entered. In this phase, subjects were simultaneously presented with two targets. The conditioned scents had been applied to these targets such that, for each subject, one target was scented with the CS+ while the other was scented with the CS-. (These target conditions will be referred to as T+ and T- respectively.) Aggressive responses to T+ and T- were recorded both within and across sessions. For purposes of analysis, the within session factor consisted of half session blocks of trials, and was included insofar as it was felt that whatever discriminative attacking may occur might extinguish early within sessions.

Method

Subjects

The subjects were 18 Long-Evan's strain male hooded rats. They were 220 days old when the experiment commenced, and were chosen from a colony of rats that had been individually caged from 34 days of age. Each subject had been handled at least once per week up to the commencement of the experiment.

Apparatus

The experimental chamber consisted of a modified Grason-Stadler rat station, model E3125C-100. It measured 9" wide x 11 3/4" in length x 8" high from grid floor to ceiling. A downward extension added to the ceiling (see Illustrations 1 and 2) effectively lowered much of the chamber's interior height to 7". The front and back walls as well as the top (including the downward extension) were constructed of clear plexiglass enabling an unobstructed view of the interior. The chamber's remaining two walls were metal. The grid floor was constructed of 18 3/16" diameter, stainless steel rods spaced 3/8" apart. Scrambled electric shock could be delivered through these grids as well as through the chamber's two metal walls.

Illustration 1 depicts the chamber as used for the training phase. The back wall was 3/8" thick and contained a circular aperture, the center of which was 1 3/4" from the grid floor. The aperture was bevelled from an interior diameter of 1 1/4" to an exterior diameter of 1". The rat, by placing his nose into the aperture, was able to reach a steel spout from which .4% saccharine solution could be attained. [This intensity of saccharine solution has proven effective in eliciting high rates of drinking in water-deprived rats (Wood, Note 2).]

Illustration 1: Experimental chamber during training phase

Photocells were placed in holders on the exterior side of the wall and positioned on either side of the aperture. When a rat placed his nose into the aperture to drink, he effectively broke the photocell beam. Electronic clock-counters automatically recorded the total time per session that the beam was broken. Similar timers, activated manually by depressing a microswitch, were used to record observations of the total time per session that each subject groomed.

Illustration 2 depicts the chamber as remodified for the test phase. The back wall was replaced with a solid panel of plexiglass. The two target models were placed in the right and left back corners, and were positioned to approximately face the front-centre area of the chamber. Directly in front of each target was a cage-like arrangement consisting of six grids rising vertically from a small plexiglass base attached to the floor grids in such manner that each cage grid was in contact with a corresponding floor grid. This arrangement enabled shock to pass through the cage grids as well as through the floor grids. Two of the cage grids were bent and their height allowed the target's head and upper torso to extend out of the cage. The four other cage grids were embedded in another plexiglass holder at ceiling height. The whole arrangement was designed to prevent subjects from escaping shock stimulation by getting behind and/or climbing on top of the nonconducting target.

The experimental chamber was enclosed in a Lehigh Valley sound-attenuating shell with interior dimensions of 28" x 28" x 31 1/2" high. A 100 watt incandescent lamp was used to light the interior. The shell's one-way observation window allowed an unobstructed view of

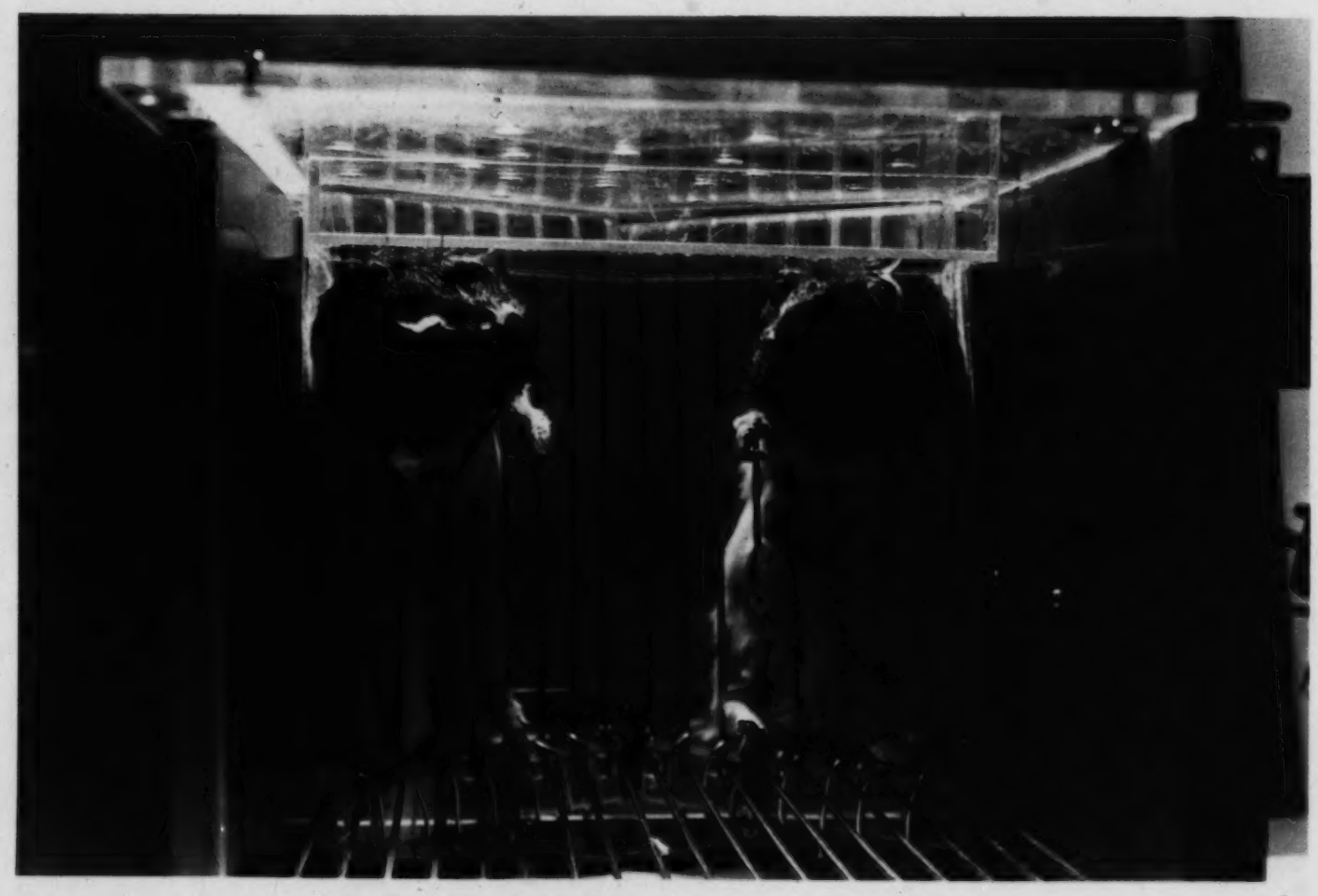

Illustration 2: Experimental chamber during test phase

the chamber inside. White noise of 68 db intensity was delivered to the shell's interior to further mask extraneous noise.

A BRS shock generator-scrambler, model SGS-003, was used to deliver the scrambled shock stimulus. The delivery of the intermittent shock stimulation was controlled and timed by Grason-Stadler and Lehigh Valley timing equipment.

For test sessions, an Esterline-Angus multi-event recorder, the pins of which could be activated manually by depressing microswitches, was used to record the observations of aggressive behavior.

Scent stimuli consisted of Heinz brand white vinegar and 50% Pine-Sol solution.[1] A fine mist spray applicator was used to apply the scent treatments.

Targets were made from study-skins of recently terminated male hooded rats. Torso stuffing consisted of cotton batton wrapped tightly around a 7" stick, which served as a support to keep the target erect when placed upright. Wire slipped into the front and back legs enabled these appendages to be manipulated. Target heads were stuffed with cotton batton, and the mouths were sewn shut. The targets were designed and positioned in the chamber to look as much as possible like live rats standing upright with front legs extended. This position is

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[1]These scents are discriminable from each other for humans, and it was assumed they would be even more discriminable for rats since it has been shown that rats are sensitive to variations in olfactory stimulation (e.g., Pierson, 1974). Of particular relevance to this experiment, Tondat (1974), in a study dealing with the learned helplessness effects of repeated shock stimulation on later shock-induced aggression, used pine-sol solution as a CS to predict the occurrence or nonoccurrence of the shock UCS. Finally, it has also been shown that the frequency of agonistic behavior in rodents can be directly influenced by variations and alterations in the natural scent of the target animal (e.g., Barnett, 1963; Mugford & Nowell, 1971; Myer, 1964; Ropartz, 1968).

an approximation of the stereotyped fighting posture which previous researchers (e.g., Ulrich & Azrin, 1962) have noted to be characteristic of shock-induced aggression in rats.

Procedure

General Procedure:

Throughout the course of the experiment, subjects were on a 23 1/2 hour water deprivation schedule, receiving 1/2 hour of access to water per day in addition to any such access provided during sessions. Each subject received one session per day, and all subjects received the same type of session, e.g., CS+ or CS- during the training phase, on any particular day.[2] The first nine subjects (Group 1) were run in the morning, while the last nine (Group 2) were run in the afternoon. Subjects within each group were run in three sets of three subjects each. There was approximately a 20 minute break between each set of sessions. Immediately preceding each set, regardless of whether subjects were or were not being shocked that day, "buffer" rats were shocked. This ensured that excretory scent in the chamber was, to some extent, constant throughout all sessions, since buffer rat excretions would tend to mask any across session variations in excretory scent from preceding subjects. Subject variations may have correlated with whether CS+ or CS- sessions were being given on a particular day, thereby possibly interfering with, and attenuating conditioning to,

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[2]It has been shown that conditioning can occur despite long delays between stimulus presentations. Baker (1977) successfully achieved conditioned inhibition in rats by using a 24-hour delay between CS and UCS presentations.

the intended scent stimuli. To further reduce such a possibility, following each session, the paper towels lining the bottom tray were replaced, and the chamber itself was washed, dried, and aired.

Training Procedure:

Training commenced with a series of consecutive non-shock sessions to allow subjects to adapt to the experimental chamber. Neither of the scent stimuli to be conditioned was present during these sessions. Degree of adaptation was determined on the basis of the total amount of time per session that each subject engaged in drinking the available saccharine solution. After 16 sessions, drinking rates had stabilized at a relatively high level, and the conditioning sessions were begun.

Conditioning involved exposing subjects to CS+ sessions interspersed with CS- sessions. Preceding each session, the appropriate scent stimulus was applied to the paper towels used to line the bottom tray. The scent was sprayed evenly over the surface of the paper until it was fairly damp, and an attempt was made to ensure relative consistency of stimulus intensity across sessions. The scent stimuli were counterbalanced between groups such that, for Group 1, vinegar served as the CS+ and pine-sol served as the CS-, whereas, for Group 2, pine-sol served as the CS+ and vinegar served as the CS-.

All sessions commenced with a 5 minute CS presentation. For CS- sessions, the subject was removed from the chamber following the 5 minute exposure period. For CS+ sessions, the exposure period was immediately followed by the UCS, consisting of 10 scrambled shocks of .5 sec duration and 2.0 ma intensity with an intertrial interval of 2 seconds.

Approximately twice as many CS- sessions than CS+ sessions were

conducted. Pilot data had indicated the possibility of a severe lag in CS- as opposed to CS+ conditioning, and it was hoped that the extra CS- presentations would compensate for such an effect.

Conditioning began with a CS+ session followed by two CS- sessions. This was followed by three sets of three CS+ and six CS- sessions each. With each set, the CS+ and CS- sessions were partially randomized, the constraints being that there could be no more than two consecutive CS+ sessions nor 3 consecutive CS- sessions. However, because the final set ended with three consecutive CS- sessions, an extra CS+ session was added to facilitate determination of conditioning level by comparison of this session with the session preceding it. The final set, therefore, essentially consisted of six CS- and four CS+ sessions.

Three dependent measures were utilized to index level of conditioning:

1. Drinking Rate: This measure consisted of the total time per session that each subject engaged in consumption of the saccharine solution during the CS period.
2. Grooming Rate: Observations were recorded of the total time per session that subjects groomed during the CS period.
3. Defecation Rate: This measure consisted of the number of separate boli per session that each subject defecated during the CS period. To ensure equal observational conditions between CS+ and CS- sessions, only those boli which could be viewed from the observation window were counted.

Only for the last set of conditioning sessions were accurate observations made of grooming and defecation behaviors. The test phase

was entered when these two measures had indicated that subjects had discriminatively conditioned to the CS+ and CS-.

Test Procedure:

The test phase involved shocking subjects in the presence of the T+ and T- targets, and recording the occurrence of aggressive behavior as well as the target toward which such behaviors were directed. Each subject was given a total of four test sessions at one session per day.

Targets were positioned in the chamber as described in the apparatus section. The targets were sprayed with the appropriate scents, and following each set of three subjects the scent was reapplied. The positions of the T+ and T- targets were alternated each test day, as well as being counterbalanced between groups. After the first two days of testing, a new pair of targets was used as the original pair had incurred considerable damage.

Each session commenced with a one minute nonshock period to allow subjects some opportunity to localize the scented targets. This was followed by 50 scrambled shocks of .5 sec duration and 5.0 ma intensity, with an intertrial interval of 2 seconds. It should be noted that on the first test day, subjects did not seem as reactive as pilot testing had indicated they would be to the 5 ma shock. It was determined that the presence of the targets had increased the amount of resistance in the chamber, thereby attenuating the shock intensity subjects received. Tape and cardboard were then used to minimize the amount of contact between the targets and the chamber's grids and steel walls. This appeared to alleviate the problem for the remainder of testing.

An observer who was blind as to which target was T+ or T- recorded number of attacks and postures to each target. An attack was defined

as a subject contacting a target by biting it or striking at it with extended forepaws. The attempt was made to eliminate contact that occurred because of the subject's random thrashing about during shock stimulation. A posture was recorded when the subject did not make aggressive contact with the target, but it appeared as though an attack was about to occur, i.e., the subject stood on hind legs facing the target with mouth open as though about to bite. As a reliability check, an extra (but nonblind) observer independently recorded aggressive behaviors for six randomly selected sessions each day.

Animal Welfare

The present research was presented to and approved by the Bioscience Animal Policy Committee. The shock parameters chosen match those reported in other pain-aggression studies with rats, and have been found relatively safe for the animals involved. Further, subject rats were weighed intermittently to ensure that the experimental manipulations were not having adverse physiological effects as evidenced by severe weight loss. No such evidence was found during the course of the experiment.

Results

Training Phase

1. Drinking Rate: By the 16th day of adaptation, the mean drinking rate per subject was 251 seconds out of a total possible of 300 seconds (5 minutes) per session. For the session following the first CS+ session, the mean had decreased to 19 seconds per subject. By the ninth session of conditioning, the drinking rate was essentially nil, and it remained at this level for the remainder of both CS+ and CS- sessions. Because drinking behavior did not recover, observations of grooming and defecation behavior were undertaken for the last set of conditioning sessions.
2. Grooming Rate: For the last set of conditioning sessions, grooming was not displayed in the presence of the CS+, but it was displayed, by 7 of the 18 subjects, in the presence of the CS-. Six of these "groomers" were in Group 2, and they accounted for 98% of the total 523 seconds of grooming observed. During the last CS- session of the set, these six subjects groomed for an average of 19.7 seconds per subject with a range of 2.4 to 34.3 seconds. Subject #8 of Group 1 groomed for a total of 9.1 seconds for the first two CS- sessions of the set, but this behavior then ceased for the rest of the conditioning sessions.
3. Defecation Rate: (To facilitate analysis, two of the intermediate CS- sessions were randomly eliminated to attain equality between treatment cells. The first and last CS- sessions were retained to ensure adequate representation and comparison of rates at the start and the end of the set.) Subjects defecated more boli in the presence of the CS- ($\underline{M}$=4.07) than in the presence of the CS+

(M=2.86). This difference was significant, $F(1,16)=8.29$, $p<.025$. In addition, Group 2 defecated significantly less boli than Group 1 (Ms=2.85 versus 4.08: $F(1,16)=9.45$, $p<.01$).

Test Phase

Three subjects from Group 1 and one subject from Group 2 displayed no aggression toward the targets during any of the test sessions. These subjects were eliminated from further analysis.

Analysis of the data for reliability sessions revealed that the two observers were in approximately 70% agreement concerning occurrence (and 94% agreement concerning nonoccurrence) of attacks, but 0% agreement concerning occurrence of postures. Reliability for attack data was considered adequate, but posture data was not included in any of the following analyses.

Attack data was subjected to a reciprocal transformation (1/x+1) to remove mean-variance dependency (Kirk, 1968). This transformed data was used in all of the following computations, tables, and graphs.

It should here be noted that attacks to the two targets were treated as independent rather than dependent. This seemed valid insofar as the occurrence of aggression did not seem to involve the painfully stimulated animal "choosing" between the two targets. Rather, subjects seemed to aggress only when they had incidentally come upon a target during the process of scrambling about the chamber. In other words, the situation seemed to amount to random presentations of T+ or T- with the possibility of attack dependent upon such presentation.

The overall analysis of variance revealed a nonsignificant Target main effect, i.e., frequency of attacks to T+ versus T-, $F<1$. Group and Half-session main effects were also nonsignificant, $Fs<1$. However,

the Session main effect approached significance, $F(3,180)=2.55$, $p<.10$ (see Figure 1), as did the Group x Target x Session interaction, $F(3,180)=2.36$, $p<.10$ (see Figure 2).

A trend analysis of the Session main effect revealed a significant Quadratic component, i.e., an increase followed by a decrease in rate of attacks, $F(1,180)=7.09$, $p<.01$. However, because of the shock intensity problems encountered during the first day of testing, the trend effect was reanalyzed for the last three sessions only. It was thought that this might yield a more valid indication of the Session effect, since shock intensity would be more consistent across these three sessions. This reanalysis resulted in a significant Sessions effect, $F(2,180)=3.46$, $p<.05$, with a significant Linear component, $F(1,180)=5.04$, $p<.025$. In other words, across the final three sessions, there was a significant linear decrease in rate of aggression.

Using F-tests for Simple Effects, a within analysis of the Group x Target x Session interaction disclosed a significant Target x Session interaction for Group 2, $F(3,180)=2.73$, $p<.05$. This contained a significant Target x Linear component, $F(1,180)=6.40$, $p<.025$. In other words, for Group 2, the across sessions decrease in frequency of attacks to T+ differed significantly from the across sessions increase in frequency of attacks to T-.

Finally, a Duncan's Multiple Range Test was carried out on the means for the Target x Session x Half-session interaction since it seemed possible that if a Target effect did exist, it may have extinguished early within the first one or two sessions. Only one meaningful comparison approached significance, that being the greater frequency of attacks to T+ than to T- for the first half of the first session,

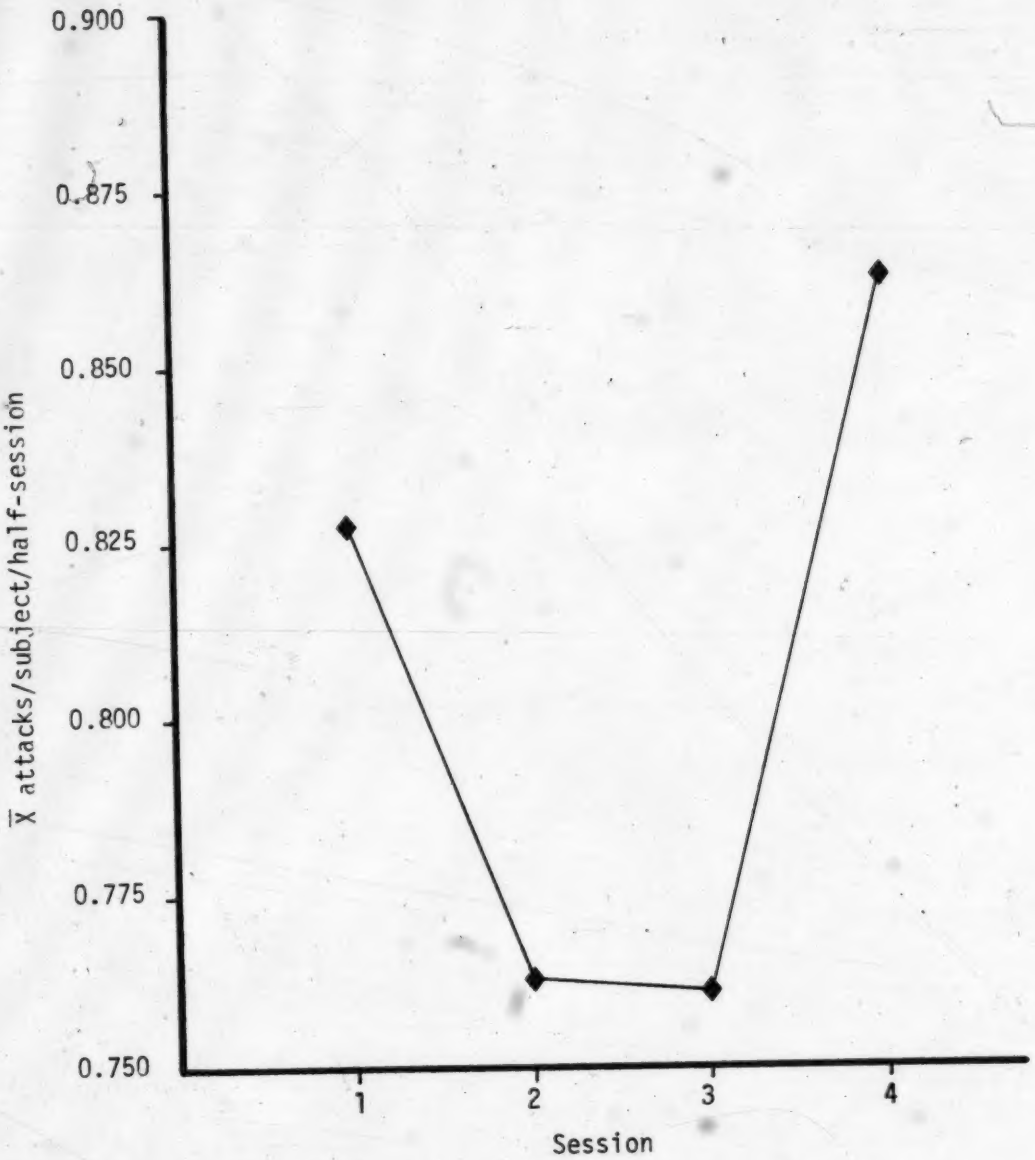

Figure 1: Session Main Effect (data transformed 1/x+1)

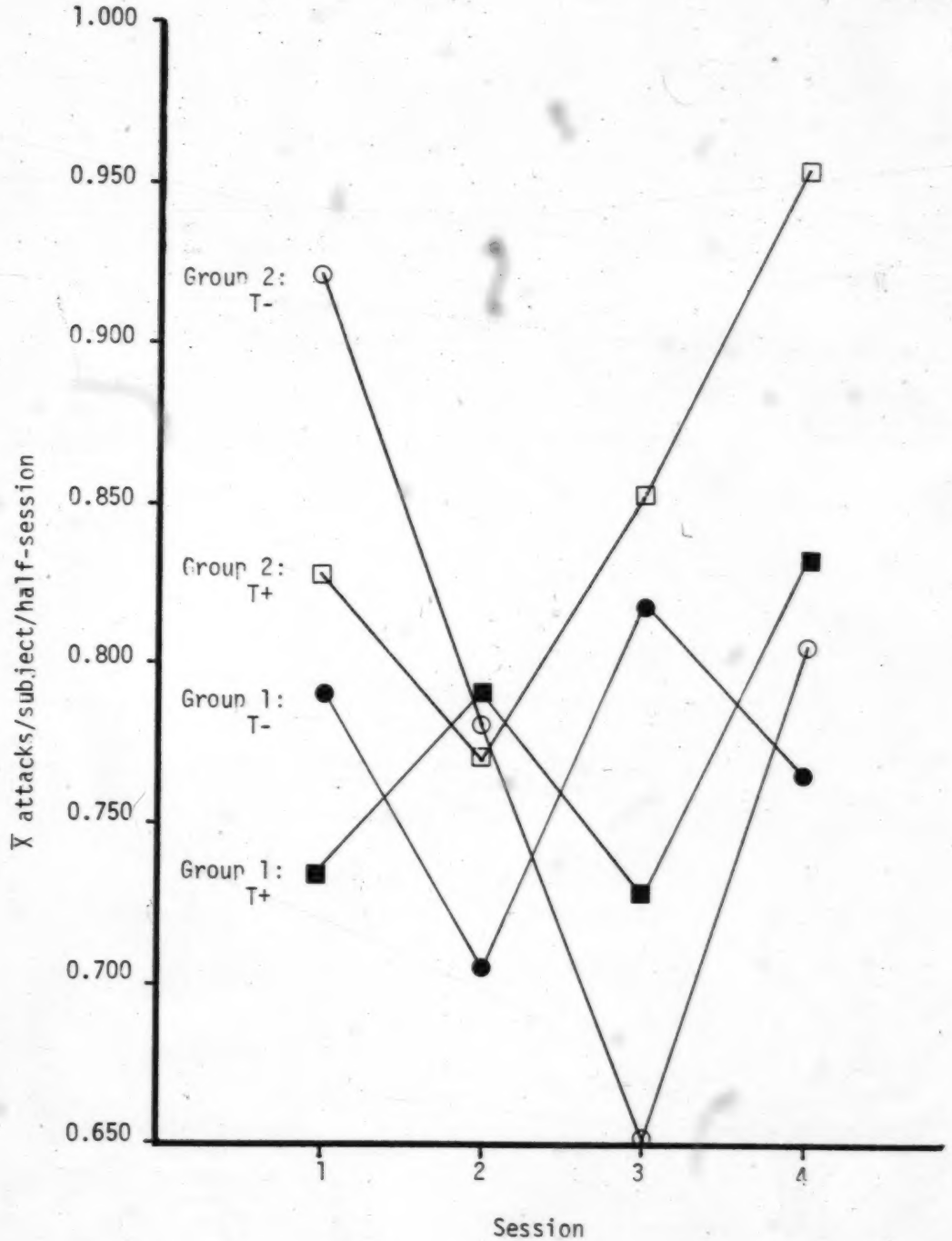

Figure 2: Group x Target x Session Interaction (data transformed 1/x+1)

$p < .10$ (see Table 1). A similar analysis of the four-way Group x Target x Session x Half-session interaction revealed no meaningful comparisons even approaching significance.

TABLE 1

TARGET x SESSION x HALF-SESSION INTERACTION
(data transformed 1/x+1)

Session	Half-Session	T+	T-
1	1st half	.691	.893
	2nd half	.886	.839
2	1st half	.816	.774
	2nd half	.740	.723
3	1st half	.771	.708
	2nd half	.827	.736
4	1st half	.851	.848
	2nd half	.952	.800

Note: Difference between means for the first half of the first session approaches significance, $p<.10$; Duncan's Multiple Range Test.

Discussion

Training Phase

While certain measures did indicate that subjects had achieved some level of discrimination between the CS+ and CS-, it must be conceded that conditioning was minimal. More specifically, the fact that the drinking response was completely eliminated, and never recovered even in the presence of the CS-, indicated that the chamber itself may have been functioning as a conditioned aversive stimulus. Another possibility is that strong conditioning to the CS+ resulted in generalization of excitatory conditioning to other strong scents, i.e., the CS-. Either way, it seems reasonable to assume that the appetitive drinking response is, to at least a certain extent, dependent on a nonaversive environment, a condition which the CS- sessions did not meet.

At least part of the problem may have been due to a lag in CS- conditioning. Because this possibility was foreseen, extra CS- presentations were given to prevent it. Although this may not have been sufficient, we may assume that continued conditioning sessions would have eventually overcome the problem. However, since it was impossible to judge when, or whether, this eventuality would occur, it was decided that the test phase should be entered as soon as some evidence of conditioning to the scents could be demonstrated. The grooming and defecation measures did provide such evidence.

The grooming measure, which revealed that seven of the subjects groomed only during the CS- as opposed to the CS+, provided evidence that at least some subjects were discriminating the two scents. However, insofar as six of these subjects were from Group 2, some interpretation of such a strong group difference is required. Two possible

explanations concern the scent counterbalancing between groups. For Group 1, the CS+ was vinegar while the CS- was pine-sol. For Group 2, the opposite combination was used. One possibility is that pine-sol as opposed to vinegar in some manner inhibits the grooming response, perhaps because pine-sol may be a somewhat aversive scent to the rat. Although this explanation is highly speculative, it implies that the group difference may not be related to a difference in level of conditioning. However, another possible explanation is that the group difference occurred because pine-sol can be more effectively conditioned as a CS+ than as a CS-, and/or vinegar can be more effectively conditioned as a CS- than as a CS+. In other words, there may be some stimulus specificity variable involved, where one stimulus is more easily conditioned as a CS for a certain UCS than is another stimulus. This effect has been shown, for example, in taste aversion studies (e.g., Revusky & Garcia, 1970). In the present situation, the rats may have found the smell of pine-sol more aversive than the smell of vinegar, thereby in some way making it easier to condition the pine-sol as the predictor of the aversive shock UCS. Similarly, the less aversive vinegar stimulus would more easily have been conditioned as the predictor of nonshock. On the basis of this reasoning, it therefore seems possible that the group difference could be attributed to a higher level of discriminative conditioning by Group 2 than Group 1.

The defecation measure also indicated that subjects were capable of discriminating the CS+ from the CS-. However, results here were in the opposite direction to that normally expected with subjects defecating significantly less boli during the CS- than the CS+. It is generally assumed that the number of boli rats defecate is in direct proportion

to the aversiveness of the situation they are in and their resultant anxiety level (e.g., Hall, 1938). However, this viewpoint has not remained unchallenged. After an extensive series of studies attempting to equate "emotionality" and anxiety with defecatory reactivity, Tobach and Schneirla (1962) conclude that "reactivity is likely to increase or decrease according to the nature of the disturbing situation and according to the given individual's developmental history ..." (p.229). Further, the present experiment is rather atypical of most studies dealing with eliminative responses in that subjects were water deprived. This may have created a certain physiological state that resulted in an inverse stress-defecation relationship. Another variable to note is that the measure used was number of boli defecated which may not necessarily correlate with total amount of faecal matter defecated. It is feasible that larger boli could have been excreted during the high stress CS+ sessions, such that, while number of boli decreased, total amount of faecal matter defecated could have remained steady or even increased.

As with grooming, the defecation measure also revealed significant group differences with Group 2 defecating significantly less boli than Group 1. On the basis of our previous reasoning for the CS+ versus CS- difference, in which it was speculated that there may have been an inverse stress-defecation relationship, we might conclude that Group 2 experienced greater overall stress than Group 1 throughout training. This, however, seems rather difficult to explain, and a more reasonable explanation might be to interpret the difference on the basis of differential level of conditioning between groups. As previously discussed, the grooming measure suggests such a difference with Group

2 demonstrating better conditioning. In the present case, for the group difference to reflect better conditioning by Group 2, we would have to hypothesize two separate stress-defecation relationships. An inverse relationship would exist during CS+ sessions such that the better conditioned Group 2 would experience greater stress resulting in less boli. A direct relationship would exist during CS- sessions such that the better conditioned Group 2 would experience less stress again resulting in less boli. Given the conclusion of Tobach and Schneirla that the stress-defecation relationship is by no means clear-cut, this present speculation is reasonable. In fact, if we imagine the stress-defecation relationship in the present experiment to be an inverted U-shaped function, then it is quite possible to derive the present group difference as well as to incorporate the inverse relationship found for the CS+ versus CS- treatments within subjects. Figure 3 depicts the situation where both groups defecate less during the CS+ than the CS-, in which Group 2 defecates less than Group 1 during the CS+, and in which Group 2 also defecates less than Group 1 during the CS-. In summary, the argument being made here is that the group difference for rate of defecation, as with grooming rate, may be indicative of between group differences in level of conditioning. This conclusion is speculative but it is important insofar as it has relevance for the discussion of some group differences found in the test results.

Test Phase

In general, the test results did not support the main hypothesis that the T+ target would be attacked significantly more often than T- target. However, some tendencies in the predicted direction were noted, and these will now be discussed and interpreted.

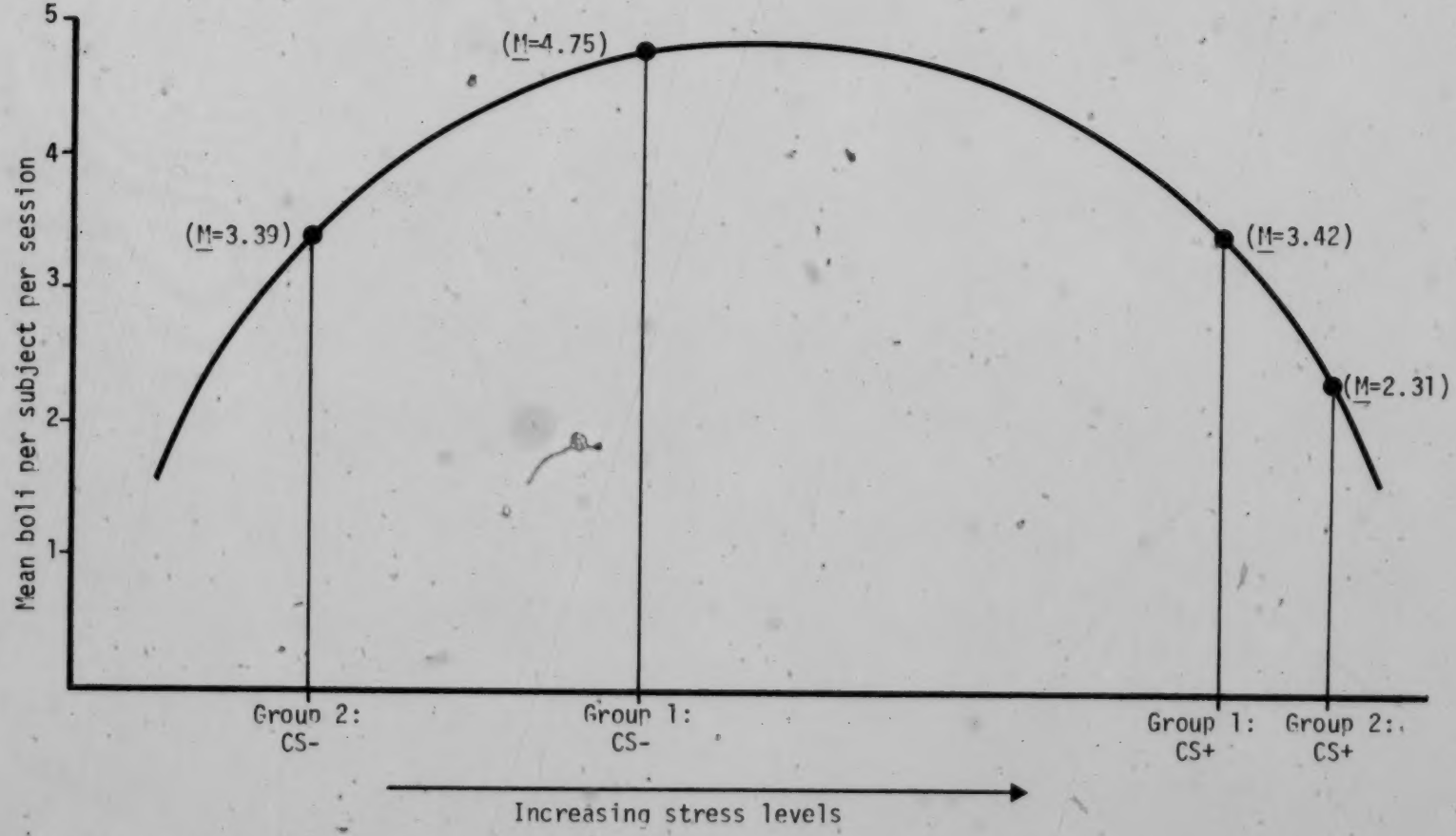

Figure 3: A conceptualization of training phase group and treatment defecation differences explained by stress levels due to conditioning.

One result that tended to support the hypothesis, though significant levels were not reached, was the Target x Session X Half-session interaction. For the first half of the first session, frequency of attacks to T+ exceeded that to T- to near significant levels, $p<.10$. It had been previously conjectured that, if differential attacking did occur, it might extinguish early within the test session. The present results support this contention, and there are a number of possible explanations as to why, if this was the case, significant results were still not attained. First, scent conditioning was not optimal as indicated by the failure of subjects to resume drinking in the presence of the CS- by the end of the training phase. This problem may have been further compounded by the fact that scent presentations of the test phase were by no means identical to the scent presentations of the training phase. During the test phase, the vinegar and pine-sol smells were confounded with the inherent smell of the targets to which they were applied. We may assume that, at best, conditioning was at least somewhat attenuated by the necessity of subjects to generalize their training experience to the confounded test situation. Another problem was the fact that shock intensity seemed somewhat attenuated during the first day of testing. If shock problems had not been encountered, attack frequency may have been higher for the first session, and any tendency to differentially attack the targets might have become more clearly apparent.

Another finding of interest was that for Group 2 there was a significant Target x Linear interaction consisting of a decrease in attacks to T+ concomitant with an increase in attacks to T- across sessions. In the discussion of the training phase, the argument was

presented that the group differences found for the grooming and defecation measures may have been due to better conditioning by Group 2 than Group 1. This possibility lends additional credibility to the notion that the present interaction was the result of previous scent conditioning insofar as it was demonstrated by Group 2 only. However, the interaction in and of itself is not so easily interpreted. It may be that subjects initially tended to attack the T+ because of its scent association with the UCS, but as testing proceeded and the shock continued unabated, contact with the T+ was avoided instead.

In other words, when aggression proved unsuccessful in removing the painful stimulation, the alternative tendency to escape, or at least to avoid the T+ may have become predominant. It may be that, given a situation where aggression results in no positive outcome, a tactical switch to escape or avoidance of an aversive target may be an adaptive response tendency in terms of natural survival. It should also be noted that escape behavior can effectively compete with and attenuate the frequency of shock-induced aggression (e.g., Azrin, Hutchinson, & Hake, 1967). However, how can the concomitant increase in attacks to T- be accounted for? A possible answer is that one function of shock-induced aggression may be to at least partially relieve increasing levels of pain-induced stress. Thus, if attacks to T+ are inhibited, stress will not be relieved, and aggression to the alternative target, T-, will be more likely, assuming a direct relationship between level of stress and probability of attack. That pain-induced stress may be reduced by aggression is supported by the research of Weiss, et.al. (1967). They found that rats given the opportunity to aggress in a shock situation were less likely to develop stress-related gastric lesions than

rats not given such opportunity.

In summary, the above discussion leads to the conclusion that, at least in the early part of the testing phase, the subjects, particularly those of Group 2, may have been attacking the T+ target on the basis of its scent association with prior shock stimulation. Perhaps further experimentation with reliable and optimal shock intensity throughout testing, as well as perhaps a somewhat modified design to optimize the level of conditioned discriminability between the targets, would result in a more adequate test of the hypothesis.

The present experiment also tested two minor hypotheses of an empirical nature. The first of these, that rats will attack inanimate targets, was well-supported by the present experiment. During testing, 15 of the 18 subjects exhibited attack behavior to such targets. In addition, reliability data indicated near 70% agreement between two observers as to the occurrence of such attacks, which is a fair, if not optimal, level of agreement.

As previously mentioned, Ulrich and Azrin (1962) concluded that rats will not attack inanimate targets, the result being that most studies since that time have employed live targets. However, a re-examination of their article reveals that they used the Holtzman Sprague-Dawley strain because these rats are relatively docile. Assuming that it was this docile strain they employed while testing for aggression to inanimate targets (this is implied, though not explicitly stated, in their article), the docility factor may have accounted for their results. The present study employed the Long-Evans hooded strain which are noted for relatively high activity and aggressiveness. It seems possible therefore that the results attained in this study may

be applicable to this strain or other similarly aggressive strains, and that similar results would not be attained with any of the more passive strains. A study directly comparing docile versus aggressive strains would be necessary before any solid conclusions could be drawn.

The other major hypothesis tested was that, given inanimate targets, subjects would exhibit a decreasing rate of attack across sessions. This hypothesis was also confirmed with such a tendency being significantly exhibited across the last three test sessions. This result, along with similar results from pilot data, also tends to confirm the notion that UCS intensity was somewhat attentuated during the first day of testing, and that the relatively low rate of attack exhibited on that day should be attributed to that attenuation.

It has been generally conceded that one characteristic of shock-induced aggression is its persistence across sessions (e.g., Ulrich & Azrin, 1962; Ulrich & Craine, 1964). On the basis of the present result, it appears that this persistence may be due more to the reactivity of the live target than to shock itself. With a reactive target, once shock has induced some attack behavior such behavior is perhaps more likely to be maintained insofar as each rat will view its opponent as a potential attacker. Meyer (1969) suggests that aversive stimulation is likely to motivate rats to engage in behaviors toward which they are so predisposed in a given situation. Two live rats in a small chamber are most likely to be predisposed to engage in agonistic behaviors. We could expand this supposition somewhat by suggesting that once rats have attacked each other they will be even more predisposed to engage in such behaviors. Thus, given a reactive adversary, a high

rate of attack can be maintained across successive sessions. With inanimate targets, however, the subject may not only have less of an initial predisposition to attack such a target, as opposed to a live active one, but the inanimate target will also not retaliate thereby not providing any basis for the maintenance of the agonistic predisposition.

Of course, another possible explanation would be that live targets are in some manner reinforcing to attack despite the possibility of aversive retaliation from such targets. Insofar as attacks function as a means of destroying, removing, or establishing dominance over a target, it seems reasonable that a target which does not provide any reactive feedback would most likely be a dead or defeated target. Since it would be highly inefficient for animals to continue attacks toward such targets, the reactivity of a target to an attack may be an inherently necessary reinforcer or cue for the maintenance of such attacks. For example, Barnett (1963, p.90) suggests that target movement is an important stimulus condition for the maintenance of attack in naturally-occurring agonistic encounters between wild rats. A comparative study utilizing targets which varied in reactivity to shock-induced attack would provide some indication as to the validity of this hypothesis for shock-induced aggression.

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APPENDIX 1

ANALYSIS OF VARIANCE SUMMARY TABLE FOR TRAINING PHASE

RATE OF DEFECATION

Source	df	MS	F	p<
Group (G)	1	55.01	9.45	.025
$Error_1$	16	5.82		
CS treatment (CS)	1	52.57	8.29	.005
Session (S)	3	5.23	1	ns
G x CS	1	.56	1	ns
G x S	3	7.54	1.19	ns
CS x S	3	8.17	1.29	ns
G x CS x S	3	4.29	1	ns
$Error_2$	112	6.34		

APPENDIX 2A

ANALYSIS OF VARIANCE SUMMARY TABLE FOR TEST PHASE

RATE OF ATTACK

(data transformed 1/x+1)

Source	df	MS	F	p<
Group (G)	1	.09	1	ns
$Error_1$	12	.78		
Target (T)	1	.05	1	ns
Session (S)	3	.14	2.55	.10
Half-Session (H)	1	.02	1	ns
G x T	1	.08	1.45	ns
G x S	3	.04	1	ns
G x H	1	.01	1	ns
T x S	3	.07	1.27	ns
T x H	1	.14	2.55	ns
S x H	3	.05	1	ns
G x T x S	3	.13	2.36	.10
G x T x H	1	.04	1	ns
G x S x H	3	.03	1	ns
T x S x H	3	.05	1	ns
G x T x S x H	3	.05	1	ns
$Error_2$	180	.06		

APPENDIX 2B

SUMMARY TABLES FOR TEST PHASE SESSION EFFECT
(data transformed 1/x+1)

Trend Analysis for all Four Sessions

Source	df	MS	F	p<
Linear	1	.03	1	ns
Quadratic	1	.39	7.09	.01
Cubic	1	.01	1	ns
Error	180	.06		

Session Main Effect after Re-analysis with Last Three Sessions Only

Source	df	MS	F	p<
Session	2	.19	3.46	.05
Error	132	.06		

Trend Analysis for Last Three Sessions Only

Source	df	MS	F	p<
Linear	1	.28	5.04	.025
Quadratic	1	.10	1.87	ns
Error	136	.06		

APPENDIX 2C

SUMMARY TABLES FOR TEST PHASE GROUP x TARGET x SESSION INTERACTION (data transformed 1/x+1)

Target x Session Interaction Within each Group using F-tests for Simple Effects

Source		df	MS	F	p<
Group 1:	Target x Session	3	.04	1	ns
Group 2:	Target x Session	3	.15	2.73	.05
	Error	180	.06		

Trend Analysis of Target x Session Interaction for Group 2

Source	df	MS	F	p<
Target x Linear	1	.35	6.40	.025
Target x Quadratic	1	.04	1	ns
Error	180	.06		